COUPLED FLUID-WALL OXYGEN TRANSPORT IN ARTERIOVENOUS FISTULAE: IMPACT OF MECHANICAL STRESSES

Introduction: Neointimal hyperplasia (NIH) is an inflammatory process that affects the walls of larger blood vessels, and is a leading cause of failure of vascular interventions, including bypass grafts, arteriovenous fistulae (AVF) and arterial stents. While the exact mechanisms underlying the development of NIH are unknown, a number of studies suggest correlation with hypoxic wall regions, as well as with abnormal wall shear stress patterns. In addition, recent studies have linked mechanical wall stretching with the expression of Hypoxia-Inducible Factor-1.

Vascular interventions, such as bypass grafts and AVF can expose local vasculature to non-physiological conditions, including abnormal pressures and flow rates, which may negatively affect WSS patterns and wall oxygenation. Vessel walls are kept oxygenated by luminal blood, and from medial/adventitial vasa vasorum (VV). Previous studies speculate that impaired VV perfusion can occur in those regions where the local compressive forces exceed the VV perfusion pressure. As a result, these regions may be prone to hypoxia and development of vascular diseases such as NIH.

Methods: In the present study, we investigate this hypothesis through coupled fluid-wall oxygen transport simulations within idealised models of AVF. Initially, we perform a finite element analysis of an idealised slit-arteriotomy procedure, to characterise the mechanical stresses to which the vessels are exposed. Residual stresses are included in the analysis and the wall is modelled as a hyperelastic, multi-layered material with two families of dispersed fibres. The resulting stresses are then used to develop a model for oxygen transport, which is able to account for potential damage/hypoperfusion of the VV, as well as the effect of VV tearing due to vein mobilisation.

Results: Preliminary results show increased compressive stresses in the vein, particularly in regions subject to bending and close to the anastomosis.

Conclusion: Several studies have hypothesized a correlation between abnormal wall stresses, VV hypoperfusion and hypoxia. To our knowledge the present study is the first attempt at validating this hypothesis computationally. The results may enable quantitative comparison between the role played by luminal blood (flow regulated oxygen transport) and the VV (mechanical stress regulated oxygen transport) in controlling wall oxygenation.

More accurate characterization of the mechanical stresses and oxygen transport within the wall may help elucidate the main causes of NIH development and potentially inform techniques that abrogate the development of NIH.